

Heretofore Unrecognized Type of Pain Signals from Large Colon May Form Common Link Between Belligerent Behavior Associated with Consumption of Brown Liquors, Colic-Associated Dystemperament, Violence in ASD and Irritability in PMS

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At a time when the majority of research into the intestine-brain dynamic is centered around the emergent fields of microbiomics and the notion that many neurological symptoms may have an inflammatory trigger, this author would take the opportunity to submit for public consideration that the aforementioned trends in research may be, at times, having the effect of slowing medical progress by tempting doctors in both practical and research settings to attribute nearly all aspects of medical phenomena under the nebulous aegis of the so-called "brain-gut connection" to these previously-overlooked biological features.

At present, the latest popularly-promoted hypothesis regarding Colic-Associated Dystemperament states that the intestinal microbiome's chemical signaling may play a role in both causing the inflammation/bloating/pain associated with colic and the dystemperament that often accompanies the condition. There seems to be consensus that the condition is caused by localized gas buildup and associated bloating, however, there is no consensus beyond this. It is unknown, for instance, why it is that some children experience colic during infancy while others do not and how it is that colicky infants overcome the condition. If so, other questions include whether having had colic carries positive or negative ramifications for health later in life and what the nature of those consequences may be. For instance, appendicitis during the adolescent or adult years may be a consequence of failing to go through the rigors of colic in infancy i.e. appendicitis may be an adult manifestation of the condition known in infancy as colic. As with conditions such as Chicken Pox, the later in life the condition strikes, the stronger the immune response and the more severe the symptoms. An immune component of colic would suitably explain why symptoms are longer in duration but lower in intensity when contracted during infancy and why, if my hypothesis is correct, an initial contracture post-infancy carries far greater risk of appendicitis requiring emergency medical attention. A greater degree of inflammation brought on by a stronger immune response increases the likelihood of the deterioration of cell walls and coherent tissue structures, potentially affecting rupture of the appendix.

Central to my thinking on this matter is a chance observation made by this author of a similarity between Colic-Associated Dystemperament and anecdotal observations reported by many concerning remarkably similar effects upon mood associated with the consumption of brown liquors but not with clear liquors. These seemingly-unrelated phenomena share in common that they may be prompted by a combination of inflammation leading to intestinal constriction (in the large colon) as well as modest gas generation leading to relatively small but

consequential pockets of bloated colon. When it comes to brown liquors, tannins have been blamed both for hangovers and mood-altering effects such as dystemperament. Medical professionals have an unfortunate tendency to deny that this difference between brown and clear liquors exists at all. If asked by a patient in a clinical setting, doctors will generally respond by chastising patients for consuming alcohol whatsoever and insisting that, "all forms of alcohol make people angry." It is not clear whether this tendency has its roots in ignorance or, rather, a fear of being perceived to endorse alcohol consumption by holding one beverage type in higher esteem than another. Virtually no research has been done into this facet of the effects of alcoholic beverages; studies concerning alcohol are generally agnostic to the type of beverage being consumed. The attitude that conducting studies that discriminate between multiple types of alcoholic beverages somehow constitutes an endorsement of alcohol consumption or that there is no scientific value in more specific studies is another reality in the research community that is, in this author's opinion, forestalling progress. Linking dystemperament associated with colic with that associated with brown liquor consumption would, if successfully achieved, serve to not only prove my point but upend a series of false assumptions currently prevalent in the medical community.

It has been observed that pain and irritability go hand-in-hand, particular in the case of chronic pain. It has long been assumed that this irritability is a natural emotional response to being in constant pain. The presence of chronic pain can magnify the perceived importance of the smallest of insults. It is a fact that a person in physical pain will frequently misdirect their anger at any source of grief, however minor. Somewhere; in a part of the human brain devoid of rationality; an instinct drives people to seek to put a name and a face on the source of chronic physical pain in the hopes that they may counter and neutralize that source of pain so as to prevent further injury. Colic-Associated Dystemperament has long been attributed to an emotional reaction to chronic pain rather than what this author believes is responsible: A direct effect of a nerve signal similar to pain that has singular effects upon neural function which, although it is often sent alongside pain signals, may be conveyed to the brain even in the absence of pain signals. While it may be possible that this phenomenon can be explained by the brain gradually changing the way it handles pain signals, I would posit that the pain signals themselves morph with time due to chemical exhaustion of the mitochondria of the cells that make the electrically-active reflector molecules that, when released, cause electrical signals to be bounced back to the brain, leading to the perception of pain. Metabolic exhaustion of the nerve cells in the case of chronic pain result in shortcuts being taken in their manufacturing process, leading to structural changes to the "reflector molecules" resulting in a return signal that is qualitatively different than a traditional pain signal. These signals electrically cue one to become instantly enraged regardless of the actual duration of the pain. Pain signals originating from these "deformed reflectors," rather than having the effects associated with pain e.g. capturing attention and distracting focus away from other tasks, crying for help, and disrupting motor function to prevent further injury, instead provoke extreme anger.

This is contrary to existing doctrine that holds that either: A.) Irritability associated with intestinal inflammation is emotional or: B.) Inflammatory cytokines capable of crossing the blood-brain barrier are released from the inflamed tissues of the large colon and are responsible for brain inflammation leading to irritability. Rather, the correct answer would seem to be: C.) Signals similar to pain signals, not interpreted by the individual as pain, have an effect on neural function that causes extreme irritability. This hypothesis would seem to fit well within our understanding of “evolutionary selection” as sensing “new” pain is useful for avoiding further damage, but can inhibit performance in combat situations e.g. one may need to punch someone in the face with a broken hand, but fear of pain may inhibit this critical action. Animals may need to flee from a predator by running on an injured leg, something that would surely slow that animal down if it could not “tune out” the pain.

If a pain is perceived by the brain as an “old” or recurring pain (gas bloating of the large colon fitting this description in most cases since people experience this sort of pain repeatedly over many years of living,) it may lead to relatively modest amounts of perceived pain with disproportional changes to their temperament. In the case of brown liquors, I would suggest that the disinhibiting effects of alcohol when coupled with intestinal inflammation and gas bloating (driven by ingredients in the liquor,) results in primal, aggressive tendencies being promoted that promote survival on an evolutionary basis.

Recent studies have linked aggression in select cases of Autism Spectrum Disorder to intestinal disorders, further supporting my hypothesis. Prior to the recent study on this matter, it was said to be unknown why some children with ASD exhibited violent behavior while others did not. It was speculated (controversially) that the condition of autism, itself, underpinned the violent tendencies. I would suggest that it is my proposed heretofore unknown pain signal type coupled with disinhibition that drive that phenomenon and that ASD does not and cannot cause a person to have violent tendencies. ASD patients are simply more likely to have untreated gastro-intestinal symptoms as a result of poor communication skills and impulse control. It is the pseudo-pain signal associated with bloating that is the root cause of this aggression.

Yet another condition associated with irritability, Pre-Menstrual Syndrome, has been uniformly attributed to hormonal changes for over a half century. Given the possibility of an oblique pseudo-pain signal, even the basic medical assumptions surrounding PMS may need to be reconsidered as the uterus would undergo the same sort of chronic stretching and contracting that the intestines routinely undergo due to gas bloating. Even this seemingly-unrelated condition may, in fact, be caused by the brain’s perception that an old pain has returned. This would seem to be supported by the fact that many patients report that NSAIDs capable of blocking pain signals at the source also have the effect of reducing irritability in PMS. If PMS were hormonal, this could not be possible.

We must continually re-evaluate our assumptions in scientific matters, particularly in those matters where the science is said to be “settled.”